Sir,

Sandhu *et al.*[1] showed convincingly that oral rehydration with a glucose-polymer solution (12·5 g/100 ml, giving 730 mmol/l glucose) and 90 mmol/l sodium predisposes to hypernatraemia. In so doing they reproduced the circumstances that contributed to the epidemic of hypernatraemia in the 1950s.[2] At that time commercial oral rehydration solutions were changed to high concentrations of glucose polymers, and probably caused hypernatraemia despite an unexceptional concentration of sodium, 50 mmol/l. The imbalanced ratio of glucose to sodium, and the high concentration of the former, cause osmotic loss of water from the intestines,[2] and increase stool loss (well demonstrated in the study by Rodriguez *et al.*[3] cited also by Sandhu *et al.*).

The authors mis-stated my analysis[2] of the nutritional benefit of oral rehydration. It is not the glucose content that benefits, as they imply, but the improved appetite for *normal* foods brought about by rapid rehydration and electrolyte repletion. Trying to increase caloric density with an expensive poly-glucose is the wrong approach to nutrition as well as to rehydration.

Rapid oral rehydration with the WHO formula, using the monomer glucose, or sucrose, and early refeeding is a well-proved treatment which does not lead to hypernatraemia even in rotavirus infection.[2]

References

1 Sandhu B K, Jones B J M, Brook C G D, Silk D B A. Oral rehydration in acute infantile diarrhoea with a glucose-polymer electrolyte solution. *Arch Dis Child* 1982; **57**: 152–4.

2 Hirschhorn N. The treatment of acute diarrhea in children. An historical and physiological perspective. *Am J Clin Nutr* 1980; **33**: 637–63.

3 Rodriguez J T, Blanco R, Gray I M. Treatment of acute diarrhoea with oral electrolyte solutions (abstract). *Pediatr Res* 1978; **12**: 440.

N HIRSCHHORN
International Division,
The John Snow Public Health Group Inc,
210 Lincoln Street, Boston,
Massachusetts 02111, USA

Dr Sandhu and co-workers comment:

It is true that palatability of a glucose-polymer electrolyte solution may prove a problem in older children, but the child referred to by Dr Hughes-Davies as refusing received sufficient to allow rehydration. We agree that glucose-polymer may be incompletely absorbed as may be the case with many 'normal' often starch-containing foods, which are reintroduced immediately after rehydration. Despite evidence of malabsorption of glucose-polymer Rodriguez *et al.*[3] found recovery without hypernatraemia the rule in their subjects. However, we should like to emphasise that, contrary to suggestions in the above letters, we did not recommend the formulation used in our study for widespread use but suggested that further studies using a much lower sodium and glucose-polymer content were required. The statement by Dr Hirschhorn that a sodium content of 50 mmol/l is 'unexceptional' ignores the possible contribution of such levels to the development of hypernatraemia.

Intracranial haemorrhage due to vitamin K deficiency associated with alpha-1-antitrypsin deficiency type Pi Z

Sir,

We read with interest the report by Hope *et al.*[1] describing 3 infants with α-1-antitrypsin deficiency who developed a bleeding diathesis during the first month of life. They stressed the scarcity of such reports. We recently treated a 23-day-old infant with α-1-antitrypsin deficiency and phenotype Pi ZZ who, 12 hours after admission for evaluation of cholestasis, developed seizures, coma, bulging fontanelle, and excessive bleeding from puncture sites. A 9% reduction in haematocrit was noticed and lumbar and ventricular taps were grossly haemorrhagic. He was breast fed and had not received vitamin K at birth. The prothrombin index was low but soon increased after vitamin K administration. Shortly after the haemorrhagic episode, hydrocephalus was noted and a ventriculo-peritoneal shunt had to be implanted. At present (age 20 months) he has left hemiplegia and moderate developmental delay. Liver function tests remain only slightly abnormal.

Intracranial haemorrhage has seldom been quoted as an early complication linked to vitamin K deficiency in α-1-antitrypsin deficiency.[2] This case however, stresses the importance of this potential complication and indicates that an urgent determination of prothrombin index should be performed in any infant admitted for evaluation of neonatal cholestasis.

References

1 Hope P L, Hall M A, Millward-Sadler G H, Normand I C S. Alpha-1-antitrypsin deficiency presenting as a bleeding diathesis in the newborn. *Arch Dis Child* 1982; **57**: 68–70.

2 Sharp H L. Alpha-1-antitrypsin deficiency. In: Lebenthal E, ed. *Digestive diseases in children.* New York: Grune & Stratton, 1978: 237–42.

I FIDALGO, C VAZQUEZ, AND J RODRIGUEZ-SORIANO
Hospital Infantil de la Seguridad Social and
University School of Medicine,
Cruces, Bilbao, Spain

Alpha-1-antitrypsin deficiency, bleeding diathesis, and intracranial haemorrhage

Sir,

We should like to report another child who had α-1-antitrypsin deficiency with disordered clotting which presented as a presumed intracranial haemorrhage.

This girl was born at term to unrelated healthy parents and the perinatal period was uneventful. Vitamin K was